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Relaxin induces vascular endothelial growth factor expression and angiogenesis selectively at wound sites.

Unemori EN, Lewis M, Constant J, Arnold G, Grove BH, Normand J, Deshpande U, Salles A, Pickford LB, Erikson ME, Hunt TK, Huang X.

Connetics Corporation, Palo Alto, CA, USA.

Relaxin is a reproductive hormone that has historically been characterized as being responsible for pubic ligament loosening and cervical ripening. Recently, relaxin has been associated with neovascularization of the endometrial lining of the uterus, potentially via specific induction of vascular endothelial growth factor. Previously conducted clinical studies using partially purified porcine relaxin have described relaxin's ability to stimulate the healing of ischemic wounds, suggesting that relaxin may also have angiogenic effects at sites of ischemic wound healing. In the present study, relaxin's angiogenic effects in the context of wound repair were tested in rodent models of angiogenesis and wound healing. Relaxin showed an ability to stimulate new blood vessel formation, particularly at ischemic wound sites, and to induce both vascular endothelial growth factor and basic fibroblast growth factor specifically in cells, presumably including macrophages, collected from wound sites. Resident macrophages collected from nonwound sites, such as the lung, did not show altered expression of these cytokines following relaxin administration. Because angiogenic wound cells are frequently macrophages, THP-1 cells, a cell line of monocyte lineage that binds relaxin specifically, were tested for and shown to induce vascular endothelial growth factor and basic fibroblast growth factor in response to relaxin. In conclusion, relaxin may be useful in the treatment of ischemic wounds by stimulating angiogenesis via the induction of vascular endothelial growth factor and basic fibroblast growth factor in wound macrophages.

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Novel therapeutic strategies in scleroderma.

Denton CP, Black CM.

Centre for Rheumatology, Royal Free and University College Medical School, Royal Free Campus, London NW3, UK.

Optimal management for scleroderma (systemic sclerosis) is likely to require treatment of the underlying disease process, which remains incompletely understood, and also of the organ-based complications of this heterogeneous condition. Clinical trials evaluating several potential agents have been completed recently, including D-penicillamine and interferon alpha. Unfortunately none of these studies has suggested significant efficacy. This article focuses on new treatment approaches using existing therapeutic agents, such as prostacyclin, and considers the potential usefulness of new agents (eg, relaxin, halofuginone) or strategies such as intensive immunosuppression with peripheral stem cell rescue. Ultimately, a better understanding of disease pathogenesis may facilitate the development of targeted therapy against key events or mediators, but for the present better evaluation of existing agents and a focus on optimizing protocols for organ-based complications, such as pulmonary vascular disease or hypertensive renal crisis, are important goals.

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